II. "On the Rhythm of the Heart of the Frog, and on the Nature of the Action of the Vagus Nerve." By W. H. GASKELL, M.D. Cantab. Communicated by Dr. MICHAEL FOSTER, Sec. R.S. Received December 8, 1881.

(Abstract.)

The method of investigation employed by the author is as follows:—The heart with the vagus nerve intact having been removed from the body together with a portion of the œsophagus, a thread is tied to the very apex of the ventricle and another to the loose flap which is disclosed at the junction of the two auricles when the two aortic trunks are cut away. The piece of the œsophagus removed with the heart is held firmly in a suitable holder and the heart suspended between two horizontal levers by means of the two threads which are attached to the auricles and ventricle. Between the two levers a clamp is placed, the edges of which can be approximated to any degree by means of a fine micrometer screw; the two limbs of this clamp are placed one on each side of the suspended heart, and by means of the micrometer screw, the tissue between the two edges can be simply held firm or compressed to any extent required. In this way, with the clamp in the auriculo-ventricular groove, the beats of both auricles and ventricle are registered simultaneously and separately; the contractions of the auricles pull the upper lever downwards, those of the ventricle the lower lever upwards. Similarly by varying the position of the clamp the contractions of any two adjacent portions of the heart can be studied, as for example, sinus and auricles, base and apex of the ventricle, &c.; heat, cold, and poisons can be applied to the tissue on the one side of the clamp and not on the other; and under all these various conditions the effects of stimulation of the vagus can be observed.

The paper is divided into two parts; Part I, on the rhythm of the heart; Part II, on the action of the vagus nerve.

In Part I reasons are given for the view that discrete impulses pass from the motor ganglia to the muscular tissue, that, therefore, the normal rhythm of the heart is dependent upon rhythmical discharges from the motor ganglia, and is not due to the production by the cardiac muscle of rhythmical results from a constant stimulation. This follows from the fact that any influence which, when applied to the auricles and sinus alone, causes an alteration in the rhythm of the auricles, affects the rate of the ventricular beats synchronously; while the same influence applied to the ventricle alone, causes no alteration in the rhythm of the auricles or in the synchronism of the ventricular with the auricular beats. Thus heat applied to the ventricle alone does not alter its rhythm although it reduces the force of

its contractions; while on the other hand, when applied to the auricles and sinus alone, it quickens the rhythm most markedly. Cold, atropin, muscarin, all slow the rhythm when applied to auricles and sinus, but cause no alteration of rhythm except in extreme doses when applied to the ventricle alone.

The author then proceeds to consider the conditions which are necessary in order that each one of these impulses should produce a contraction, and concludes that a due relation must exist between the strength of the impulse and the excitability of the tissue in order to obtain this result.

By a comparison of the rate of the contractions of the auricles with those of the ventricle, it is found that the ventricle can be made to beat synchronously with every second, third, fourth, or more auricular beats, or to cease from beating altogether by increasing the compression of the clamp in the auriculo-ventricular groove or by heating the sinus and auricles alone without heating the ventricle. The commonest and most permanent effect is to make the ventricle beat synchronously with every second auricular beat.

This same want of sequence between the ventricular and auricular contractions can also be obtained by the application of various poisons to the ventricle alone. A marked difference however exists between the two cases. In the first case, when the ventricle is made to beat with half-rhythm by tightening the clamp, or by heating the auricles and sinus, its contractions are those of a strong vigorous muscle, and are more powerful than when the ventricle was beating synchronously with every beat of the auricles; on the other hand, the application of poisons to the ventricle does not produce this effect on its rhythm until by the action of the poison the force of the contractions has become greatly reduced.

For this and other reasons given in the original paper, the author concludes that either tightening the clamp or heating the auricles and sinus alone diminishes the strength of the impulses passing to the ventricular muscle, and so causes the half-rhythm observed; while various poisons applied to the ventricle alone produce the same effect by diminishing its excitability, without affecting the strength of the impulses.

The conclusions arrived at in Part I can be summed up in the following propositions:—

1. The rhythm of the heart is caused by discrete motor impulses passing to the muscular tissue from certain motor ganglia.

2. In order that each one of these impulses may produce a contraction of the ventricle a due relation must exist between the strength of the impulse and the excitability of the ventricular muscle.

3. When each impulse is inefficient to cause a contraction of the ventricle, the ventricular muscle has the power of summing up the

effects of two or more of these inefficient impulses, and so continues to beat rhythmically though no longer synchronously with every impulse.

4. The most satisfactory explanation of this summation process is as follows:—Every impulse which is inefficient to produce a muscular contraction increases the excitability of the muscle, and therefore makes it easier for a second similar impulse to cause a contraction.

5. The impulses can be made inefficient to produce contractions synchronous with them by lowering sufficiently the excitability of the ventricle, as is seen in the action of poisons, even although the rate and strength of the impulses remain unaltered.

6. The impulses can also be made inefficient, when the excitability of the muscle is unchanged, by diminishing the strength of the impulses, as is seen in the effects of compressing the tissue between the ventricle and the motor ganglia, or of heating the auricles and sinus without heating the ventricle.

7. There is a limit to the extent to which a series of inefficient impulses can raise the excitability of the muscle, so that the ventricle can remain absolutely quiescent, even although the impulses still pass to it, when those impulses are sufficiently weakened.

In Part II the action of the vagus nerve is considered, and it is shown that its stimulation produces a most marked effect upon the force of the contractions, both of auricles and ventricle, entirely independent of any alteration of rhythm. The curves obtained can be classified under the three following types:—

1. Complete quiescence of both ventricle and auricles, followed by contractions which at first are scarcely visible, but which rapidly increase in size, until at the maximum they are much greater than before the stimulation of the nerve. From this maximum they very gradually decrease, until the original size of contraction is again reached.

2. During the stimulation no quiescence of either ventricle or auricles, but simply a diminution of the size of the contractions, followed by a rapid and marked augmentation of the contractions beyond the original height, and then a slow gradual diminution to the size obtaining before the nerve was stimulated.

3. No primary diminution, but from the commencement of the stimulation the beats increase in size, and after a time gradually return again to the original size.

Between these three types every conceivable variation may occur, so that a series of curves may be selected in which no line of demarcation can be drawn between complete primary quiescence, or to use the usual term, inhibition, on the one hand, and a simple primary augmentation of the size of the contractions on the other.

These curves alone show that the vagus is able to cause a standstill

by diminishing the force of the contraction down to quiescence; this is further shown by the fact that standstill of the ventricle alone can occur while the auricles are beating with accelerated or unaltered rhythm, but diminished force, or even when from the commencement of the stimulation the force of the auricular contractions is increased.

This same gradation of effect, as the result of the stimulation of the nerve, from absolute standstill to a simple primary augmentation, is seen more or less clearly in the course of each separate experiment; the stimulations that occur immediately after the suspension of the heart are much the most likely to produce standstill; later ones to cause primary diminution followed by augmentation, and finally augmentation alone.

The power of diminishing the contractions to standstill appears to last longer after the heart has been suspended at some times of the year than at others.

The conclusion is drawn that the variations in the effects produced by stimulation of the vagus on the force of the contractions are dependent essentially upon the condition of the nutrition of the heart; and possibly for the same cause the vagus tends to lose all power of producing slowing after the heart has been suspended in the apparatus, for in most cases acceleration only is seen, although slowing occurred on stimulation before the heart was cut out, and apparently slowing is more likely to occur immediately after the suspension of the heart than later.

The action of the vagus upon the muscular tissue is not only shown by its effect on the size of the contractions, but also by its influence on the excitability and tonicity of the ventricular muscle.

When by tightening the clamp the ventricle is made to beat synchronously with every second auricular beat, stimulation of the nerve may cause the ventricle during the stimulation to beat synchronously with every third, fourth, or more auricular beats; and the same alteration in the relation between the rhythm of the two parts above and below the clamp is seen in the case of the contractions of the apex and base of the ventricle, when the clamp is placed midway across the ventricle.

Also, when the ventricle is beating with half-rhythm from the action of the clamp, stimulation of the nerve may make it beat synchronously with every beat of the auricles for a definite time; and when the ventricle is not beating, either in consequence of tightening the clamp, or of heating the auricles and sinus, then vagus stimulation may cause a series of contractions synchronous with those of the auricles.

These experiments are to be explained on the supposition that the vagus stimulation diminishes the excitability of the ventricle at one

time and increases it at another, and it is also shown that the times of this diminution and increase correspond respectively to the periods when the vagus causes a diminution and increase of the size of the contractions.

The action of the vagus upon the muscular tissue of the ventricle is further shown by its power of removing the inequalities in the size of the ventricular contractions, when as often happens, the ventricle is beating with alternately strong and weak contractions.

Stimulation of the nerve causes this inequality to disappear when it increases the force of the contractions, and to reappear again when it diminishes that force.

The effect of stimulation of the vagus upon the tonicity of the ventricle was studied by the method described elsewhere,* and the author shows that the relaxation between the beats of the ventricle is increased during the stimulation of the nerve, even although the rate of rhythm is not made slower.

The conclusion therefore is drawn, that stimulation of the vagus acts upon the muscular tissue of the ventricle in such a way as to diminish its excitability and lower its tonicity, when it reduces the force of the ventricular contractions, while it increases its excitability and possibly also increases its tonicity when it augments the contraction force.

Finally, it is shown that atropin removes the whole action of the vagus stimulation, and the effects of the local application of curare, muscarin, and atropin are described and discussed.

In conclusion, the author sums up the results of these experiments, and suggests that a series of formative processes are going on in both the muscular tissue and the motor ganglia of the heart, similar to those which occur in gland-cells, and that the vagus produces all its effects by increasing the activity of these processes and not because it contains a multiplicity of fibres, each of which possesses a different function.

## III. "On Melting Point." By Edmund J. Mills, D.Sc., F.R.S., Young Professor of Technical Chemistry in Anderson's College, Glasgow. Received December 6, 1881.

(Abstract.)

The investigation, of which the memoir contains an account, was undertaken in order to determine, with considerable accuracy, the

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